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REVIEW

SPECIAL ISSUE
THE INTEGRATIVE BIOLOGY OF THE HEART

The role of cognition as a factor regulating the diving responses of animals, including humans

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ABSTRACT

The dive response involves three main components – breath holding, reduced heart rate and increased peripheral vasoconstriction – and is ubiquitous during forced dives in air-breathing vertebrates; however, numerous studies in free-diving animals have shown that the heart rate response to diving varies considerably in a manner that suggests cognitive control. Furthermore, studies on free-diving animals and controlled experiments in trained animals both indicate that the dive response can be conditioned, such that the reduction in heart rate begins before submergence and the extent of the reduction is set early in the dive. In addition, numerous species also experience an increase in heart rate and blood flow during ascent at the end of a dive, a phenomenon commonly called 'ascent tachycardia'. Collectively, these data suggest that although the dive response is under autonomic control, many species can vary its magnitude depending on the length and type of the planned dive – an indication of a role for cognition in the overall physiological responses associated with diving. Here, we provide examples of the conditioned cardiac responses – including anticipatory changes in heart rate – in several diving species and propose potential underlying mechanisms. We also discuss how the anticipatory cardiovascular responses not only improve diving capacity, but also prevent diving-related problems, such as decompression sickness or barotrauma, through a mechanism described by the selective gas exchange hypothesis.

KEY WORDS: Diving, Breath holding, Reflex, Cognition, Bradycardia, Tachycardia, Apnoea, Ventilation, Lungs, Heart

Introduction

The cardiorespiratory changes that occur during breath-hold diving – commonly called the 'dive response' – include cessation of breathing (apnoea), a reduction in cardiac output due to bradycardia (see Glossary) and extensive peripheral vasoconstriction. Although much research has described cardiovascular changes during diving, most studies have focused heavily on the changes in heart rate and the magnitude of these changes has been used as a proxy for diving capacity.

Since the early seminal studies in both forced and freely diving mammals, it has been recognized that the dive response involves a level of anticipatory, and possibly conditioned or cognitive cardiac control (Elsner et al., 1966b; Jones et al., 1973; Kooyman and Campbell, 1972; Ridgway et al., 1975; Scholander, 1940, 1963). For example, Scholander (1963) commented that, in mammals, the dive response can be induced by a number of psychological factors, and stated that the 'bradycardia sometimes fails to develop in a submerged seal if the animal knows it is free to raise its head and breathe whenever it likes'. Kooyman and Campbell (1972) later observed in the Weddell seal (*Leptonychotes weddellii*) that there appeared to be an inverse relationship between heart rate and length of the dive (Fig. 1). They hypothesized that this relationship, as well as other observed behavioural events that appeared to be correlated with heart rate, indicated that the seals anticipate the nature of the forthcoming dive. Jones et al. (1973) made similar observations in harbour seals (*Phoca vitulina*) and this was also taken as evidence of a certain level of associative learning. In trained bottlenose dolphins (*Tursiops truncatus*) and California sea lions (*Zalophus californianus*), cardiovascular responses to diving could also be both anticipatory and conditioned (Elsner et al., 1966b; Ridgway et al., 1975), and more recent studies (described below) provide ample evidence that many species can control their heart rate during diving.

Against this backdrop, we summarize here the current knowledge on the dive response in breath-hold diving vertebrates. We suggest that although the diving bradycardia (and associated peripheral vasoconstriction and blood flow redistribution) are key components of the dive response (Burggren et al., 2024), the capacity to vary heart rate based on varying physiological needs – a capacity that includes cognitive control – is also important, and often overlooked. The ability to actively vary the cardiac response during diving, rather than relying on reflexive responses, provides a further adaptive advantage for conserving available O_2 and extending aerobic dive time. In addition, such changes also minimize the exchange of N_2 and reduce the risk of the formation of gas emboli and decompression sickness (the bends), through the selective gas exchange hypothesis, as we discuss below (Fahlman, 2024; Fahlman et al., 2021; see the following YouTube video for an explanation of this hypothesis: https://youtu.be/sfBOpUuJv1c?feature=shared).

History of the dive response

The heart rate changes associated with diving were first described by Admund Godwyn in 1786 (Vega, 2017), but it was not until 1870 that the response was more broadly publicized, when Paul Bert described how the heart rate slowed during head submersion in ducks, in what later was called the 'dive response' (Bert, 1870). In a series of studies in the 1940s to 1960s, Scholander and Irving further proposed that the dive response is an autonomic reflex, and that the resulting bradycardia and associated vasoconstriction of peripheral blood vessels act to centralize perfusion to vital organs, with little or no change in mean arterial blood pressure

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Glossary

Aerobic dive limit (ADL)
The dive duration that does not result in elevated post-dive blood lactate levels.

Accentuated antagonism
A hypothesis that there is an antagonism between the sympathetic and parasympathetic branches of the autonomic nervous system during diving.

Autonomic conflict
Simultaneous activiation of the sympathetic (cold response) and parasympathetic (diving) systems, resulting in cardiac arrythmias.

Baroreceptor reflex
Receptors in the aortic arch and carotid arteries that sense and help adjust blood pressure.

Bradycardia
A fall in heart rate.

Oxygen debt
The O_2 used from blood and muscle to fuel aerobic metabolism during a breath-hold dive.

Operant conditioning
Using reinforcement (or punishment) to alter the frequency of a behaviour.

Respiratory (central) chemoreceptor reflex
The stimulation of breathing by central chemoreceptors (cells that respond to changes in CO_2 partial pressure and pH).

Tachycardia
An increase in heart rate.

Ventilation–perfusion ratio
A ratio that describes the matching of ventilation to perfusion in the lung. Ventilation here refers to how 'filled' the alveoli are and not the act of breathing.

(Irving, 1963; Irving et al., 1941b, 1942; Scholander, 1940, 1963), as discussed below. These early diving experiments were performed by restraining animals and submerging them during 'forced' dives. This experimental design likely resulted in a high level of stress, and the cardiovascular changes – as reflected by the degree of bradycardia – under such conditions likely represent the extreme case (Scholander, 1940) (Fig. 2).

Deployment of one of the first heart rate data recording devices confirmed that the dive response in freely diving Weddell seals (Kooyman and Campbell, 1972), and gentoo (*Pygoscelis papua*) and Adélie penguins (*Pygoscelis adeliae*) is not as extreme as that measured during experimental forced dives; Figs 1 and 2). This finding was later confirmed in a number of different studies and species (Andrews et al., 1997; Fedak et al., 1988; Furilla and Jones, 1987; Goldbogen et al., 2019; Hill, 1986; Houser et al., 2010; Jones et al., 1973; McDonald et al., 2018; McDonald and Ponganis, 2014; Thompson and Fedak, 1993). The magnitude of the reduction in heart rate also varies considerably between dives in a given species, but there is a general trend for lower heart rates during longer dives (Thompson and Fedak, 1993), consistent with the notion that the degree of the response is also controlled by blood gas partial pressures (Fig. 1; Angell-James and Daly, 1969). As variation in lung volume alters cardiac acitivity (both in terms of heart rate and stroke volume in dolphins; Angell-James et al., 1981; Fahlman et al., 2023, 2019), it has also been suggested that dive depth (and lung compression) alters the magnitude of the heart rate reduction during diving (Ponganis et al., 2017). However, several studies have failed to report such a relationship (Houser et al., 2010; McDonald and Ponganis, 2014), and some studies that have reported a correlation between heart rate and dive depth do not account for the increase in dive duration associated with deeper dives. Regardless, a general reduction in heart rate during diving has been reported for both aquatic and terrestrial species, including humans (Andersen, 1961; Johansen, 1959; Scholander, 1940). These observations led Scholander to propose that the dive response is the 'master switch of life', representing a defence against asphyxia (Scholander, 1963). As data were collected from additional species, however, researchers began to comment on the lack of a strong relationship between the magnitude of the reduction in heart rate and dive duration. Moreover, even fish taken out of water and deprived of adequate gas exchange demonstrate similar cardiovascular changes (Leivestad et al., 1957). These observations led to the suggestion that the dive response might be an ancestrally conserved response to reduced access to O_2, rather than an adaptation evolved specifically for diving (Mottishaw et al., 1999). In a number of mammalian, avian and reptilian divers, heart rates during diving within the aerobic dive limit (ADL; see Glossary) are no different than heart rates at the end of an apnoeic period during intermittent breathing at the surface (Belkin, 1964; Fahlman et al., 2019; Gaunt and Gans, 1969; Kanwisher et al., 1981; Kooyman, 1985; Kooyman and

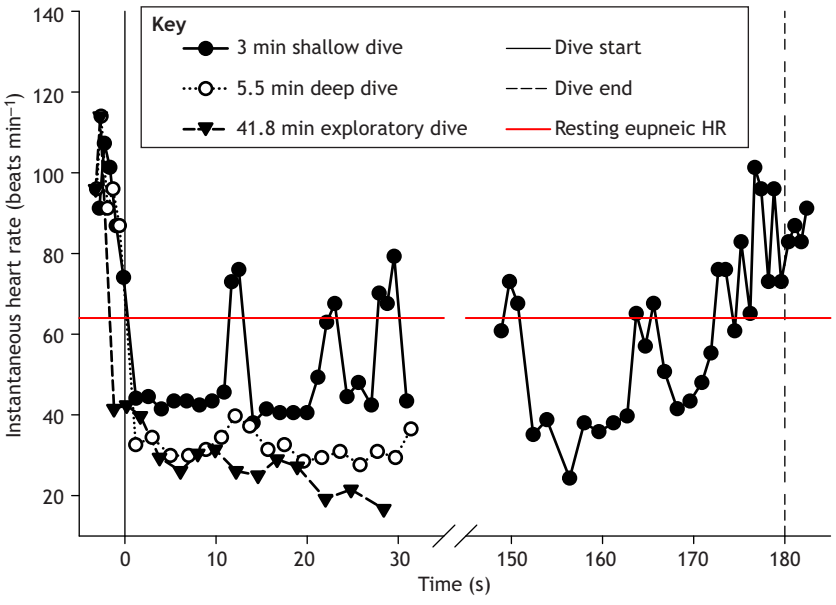

Fig. 1. Instantaneous heart rate over time in the Weddell seal (*Leptonychotes weddellii*). The figure shows heart rate (HR) before and for the first 30 s of three different types of dives, and during the last 30 s of a 3 min shallow dive. Dives are shallow, deep or long (an exploratory dive). Resting eupneic heart rate was 64 beats min^{-1} (red solid horizontal line). Body mass in adults ranged from 360 to 430 kg and the aerobic dive limit (ADL) for a 200 kg Weddell seal is approximately 15 min (Kooyman et al., 1983). The data show a distinct pre-diving anticipatory drop in heart rate (bradycardia) before the long exploratory dive and also a diving bradycardia in the deep dive. The early heart rate responses indicate that the longer dives result in a greater drop in diving heart rate. There is a distinct anticipatory pre-surface tachycardia at the end of the shallow dive. The vertical solid and broken lines are the start and end of a dive, respectively. Data redrawn from (Kooyman and Campbell, 1972).

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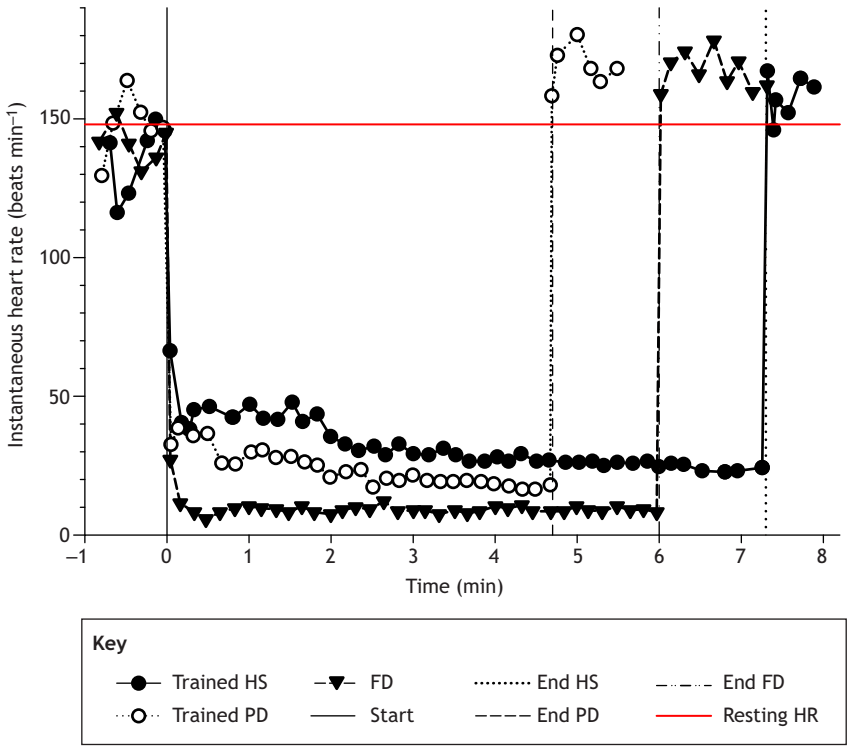

Fig. 2. Instantaneous heart rate before, during and after different forms of submergence in the harbour seal (*Phoca vitulina*). The figure shows a trained head submergence (HS), a trained pool dive (PD) and a forced dive (FD), lasting between 4.7 and 7.0 min. The solid black line is the start of the breath-hold, and the broken lines show the end of the breath-hold for each of the different types of submergence. The red solid horizontal line is the resting heart rate. The data show a much greater decrease in heart rate during the forced dive compared with either of the trained dives. Also, decrease in heart rate with dive duration is seen in the trained HS breath-hold. The calculated aerobic dive limit of a harbour seal is estimated to be around 6 min (Weingartner et al., 2012). Data redrawn from Elsner (1965).

Campbell, 1972; Lin et al., 1972). Thus, in many cases, the heart rate response that occurs during diving appears to be the result of losing the tachycardia (see Glossary) associated with respiration and not a bradycardia associated with diving (Belkin, 1964; Fahlman, 2024; Fahlman et al., 2019; Kooyman, 1985; Lin et al., 1972).

What does the dive response do?

Before discussing the role of cognition and conditioning in diving physiology, let us review the purpose of the dive response. It is essential to remember that the key controlled variable in cardiovascular reflexes is blood pressure. In the case of the dive response, the changes in heart rate simply reflect the changes necessary to maintain a constant blood pressure in the face of increased vascular resistance due to vasoconstriction. Although only a few studies have measured blood pressure during diving in mammals, pinnipeds maintain mean arterial blood pressure within a narrow range during dives (Elsner et al., 1966a; Irving et al., 1942; Sinnett et al., 1978). In cetaceans, blood pressure measurements only exist for anesthetized animals (Sommer et al., 1968). However, upregulation of *Alox5* (a gene promoting vasoconstriction through its product 5-lipoxygenase) during static breath-holds in bottlenose dolphins in managed care suggests that cetaceans also maintain stable blood pressure during diving (Blawas et al., 2021c). In humans, by contrast, the mean arterial blood pressure tends to increase substantially during diving, as well as during static breath-holds (McKnight et al., 2024), suggesting that the fall in heart rate is not sufficient to offset the increase in peripheral resistance in humans.

In forced dives, the vasoconstriction that leads to the fall in heart rate is associated with the redistribution of blood flow to different organs. In the general diving literature, it is suggested that vasoconstriction acts to maintain adequate blood flow and O_2 delivery to essential organs that are dependent on aerobic metabolism (e.g. heart and brain) by reducing blood flow to those tissues that can continue to function with a reduced O_2 supply. Scholander (1940) proposed that organs receiving reduced blood flow would: (1) be supported by endogenous O_2 stores (e.g. myoglobin stores in skeletal muscle), (2) undergo reductions in metabolism, or (3) be fuelled by anaerobic metabolism. The extent to which a redistribution of blood flow occurs, however, varies in voluntary dives in both mammals and birds (Jones et al., 1988; Zapol et al., 1979).

There is theoretical and empirical evidence that the dive response extends dive duration by reducing perfusion to muscles during both voluntary and forced dives in birds and mammals (Davis and Kanatous, 1999; Elliott et al., 2002; Scholander, 1940). In these instances, it is argued that the reduced blood flow forces muscle to use the endogenous O_2 bound to muscle myoglobin. Myoglobin has a much higher affinity for O_2 compared with haemoglobin, and only begins to release O_2 when the partial pressure in the tissue falls. Thus, despite reduced blood flow, the muscle does not necessarily become anaerobic. However, during natural voluntary dives, there is contradictory evidence that indicates that blood flow to exercising muscle is not reduced (Ridgway and Howard, 1979). In fully aquatic species, as well as in larger, semi-aquatic species, all normal physiological functions (e.g. feeding, digesting, growth, reproduction) continue during diving. This suggests that blood flow is not restricted to any organ. Some of these species may be active underwater for many minutes, thanks to a combination of large O_2 stores and very efficient means of underwater locomotion. Generally, these animals surface before their O_2 stores are depleted, quickly replenish their O_2, and dive again. However, if the dive is extended and the classical dive response occurs, then the animals will rest at the surface for much longer before submerging again (see Kooyman et al., 1980). Thus, O_2 conservation is indicated during forced dives by a profound bradycardia as blood flow is restricted to cerebral and central cardiovascular areas. By contrast, in voluntary dives, bradycardia is reduced or absent and blood flow is preferentially directed to the working muscles (Butler, 1988; Jones et al., 1988). Prolonged dives do occur naturally, of course; in these cases, blood flow redistribution and a profound bradycardia are evident (McDonald and Ponganis, 2014; Noren et al., 2012; Panneton, 2013).

We now turn to the issue of cognition and conditioning in diving animals. We use available data to support the classic argument that the cardiovascular changes associated with diving enhance diving capacity. We propose that understanding differences in the dive response lies in understanding the conditioned and/or cognitive capacity to alter the response to match the intended dive and to deal with unexpected events. Through anticipatory adjustment of perfusion early in the dive (as evidenced by anticipatory bradycardia), the diving animal can extend dive duration. Importantly, at the end of the dive, changes in perfusion (as evidenced by anticipatory tachycardia) maximize partial pressure diffusion gradients, which may act to reduce time spent at the surface by enhancing gas exchange between tissues and blood, as well as between blood and lung gas. These anticipatory changes in perfusion are part of the suite of mechanisms enabling diving animals to selectively exchange O_2 and CO_2 during the dive, all while minimizing or excluding the exchange of N_2 with the tissues (Fahlman, 2024; Fahlman et al., 2021; García-Párraga et al., 2018).

Anticipatory cardiac responses – evidence for a role of cognition

In humans and other terrestrial mammals, the capacity for anticipatory variation in heart rate appears limited, but there has long been evidence for biofeedback or operant conditioning (see Glossary) of human visceral responses (Miller, 1969; Miller and DiCara, 1967; Shearn, 1962). These studies provided evidence that conditioned control of both heart rate and blood pressure are possible (DiCara and Miller, 1968; Miller and DiCara, 1967), and may similarly exist in diving mammals and other animals. Conversely, it is possible that anticipatory control may be absent or minimal in terrestrial mammals that intermittently dive for relatively brief periods of time (e.g. muskrat, beaver, water voles, water shrews), as there may be little evolutionary pressure for such a capacity (although diving physiology generally in these terrestrial animals has received far less attention than their marine counterparts). The evidence for conditioned control of both heart rate and blood pressure is discussed in more detail below.

Anticipatory changes in diving mammals

Both pinnipeds and cetaceans show anticipatory bradycardia associated with diving (Bickett et al., 2019; Casson and Ronald, 1975; Elmegaard et al., 2016; Elsner, 1965; Elsner et al., 1966b; Fahlman et al., 2020a, 2019; Fedak and Thompson, 1993; Harrison et al., 1972; Irving et al., 1941a; Jones et al., 1973; Kaczmarek et al., 2018; McKnight et al., 2019; Noren et al., 2012; Ridgway et al., 1975). One form of anticipatory bradycardia is where the reduction in heart rate is initiated just before the animal submerges (Fig. 1). Such anticipatory bradycardia has been demonstrated in the harbour seal, where a reduction in heart rate and anticipatory changes in cerebral blood flow sometimes occur as early as 15 s before the beginning of a dive (Jones et al., 1973; McKnight et al., 2019). This suggests that these cardiovascular changes are not directly linked to submersion itself.

Anticipatory bradycardia also describes the situation where the level of diving bradycardia is set early in the dive and correlates with the upcoming dive duration. In these situations, bradycardia is not dependent on reductions in blood gas partial pressures during the dive. This phenomenon was first described in freely diving marine mammals (Kooyman and Campbell, 1972; McDonald and Ponganis, 2014; McDonald et al., 2020; Thompson and Fedak, 1993). In the Weddell seal, for example, both the rate and magnitude of the heart rate reduction are greater at the beginning of long dives compared with short dives (Kooyman and Campbell, 1972; Fig. 1). These anticipatory changes suggest that the animals have great plasticity in their cardiovascular response, allowing them to prepare to maximally conserve the available O_2 in anticipation of the upcoming dive. Similar results have been reported in trained marine mammals, where the heart rate response is related to the expected (‘anticipated’) dive duration (Bickett et al., 2019; Elmegaard et al., 2016; Elsner, 1965; Elsner et al., 1966b; Fahlman et al., 2020a; Harrison et al., 1972; Irving et al., 1941a; Jones et al., 1973; Kaczmarek et al., 2018; McKnight et al., 2019; Ridgway et al., 1975).

The question also arises as to whether the ‘decision’ to terminate a dive and return to the surface is volitional; i.e. is a cognitive decision. To date, the evidence suggests that in all diving homeotherms, most periods of submersion are short enough to be completely aerobic (Kooyman et al., 2020). Scholander (1940) stated ‘even if reduced metabolism might be thought possible during a quiet dive this cannot be the case in an ordinary dive. It is during submersion that a seal does most of its exercise in hunting fish and by distance swimming. On most occasions they surface before the oxygen stores are depleted, quickly replace the stores, and dive again’. Thus, although it seems probable that some form of chemoreceptor input underlies the decision to return to the surface, this interpretation is paradoxical, because chemoreceptor input has also been proposed to produce the bradycardia. It is also conceivable that the dive duration and the accompanying bradycardia are learned through repetitive dives (operant conditioning), particularly those undertaken by newborn animals.

Animals that are freely diving both in an experimental setting and in the wild show anticipatory tachycardia prior to surfacing (Fig. 1; Belkin, 1964; Elliott et al., 2002; Fahlman et al., 2020a; Fedak and Thompson, 1993; Fedak et al., 1988; Harrison and Tomlinson, 1960; Irving et al., 1941a; Jones et al., 1973; Kooyman and Campbell, 1972; McDonald and Ponganis, 2014; McKnight et al., 2019; Murdaugh et al., 1961; Ponganis et al., 1997b; Thompson and Fedak, 1993). Importantly, this increase in heart rate during ascent is generally gradual and not related to increasing activity associated with approaching the water surface (Fedak and Thompson, 1993; Fedak et al., 1988). Nor is this gradual increase in heart rate during ascent due to an inability to quickly elevate heart rate, because both pinnipeds and cetaceans can rapidly raise their heart rate within a few seconds while diving (Fedak et al., 1988; Houser et al., 2010). It has been proposed that the decrease in hydrostatic pressure experienced during ascent results in expansion of the gas-filled spaces and is responsible for these changes (Angell-James et al., 1981; McDonald et al., 2020). However, anticipatory tachycardia also occurs during the end of horizontal dives at shallow depth (Casson and Ronald, 1975; Elliott et al., 2002; Jones et al., 1973; McKnight et al., 2019; Signore and Jones, 1995). In the muskrat (*Ondatra zibethicus*) and harbour seal, pharmacological interventions suggest that this anticipatory reversal of the dive response is most likely due to reductions in vagal tone (Elliott et al., 2002; Signore and Jones, 1995).

The increase in heart rate during ascent has been proposed to be a mechanism to prepare the body for rapid recovery when reaching the water surface (Fedak and Thompson, 1993). Increasing heart rate, and thus cardiac output, allows the tissues to use the remaining O_2 in blood and muscle. This reduces tissue P_{O_2}, which creates a greater diffusion gradient and improves gas exchange when breathing resumes. The increased perfusion also extracts CO_2 from the tissues and brings it to the lungs, where it can be exhaled rapidly when reaching the surface. Thus, the increased perfusion creates a favourable partial pressure gradient that helps increase diffusion rates, improves gas exchange and reduces the surface recovery

duration (Fedak and Thompson, 1993). Interestingly, if the animal does not return to the surface to breathe, but unexpectedly dives down again before surfacing, the heart rate again decreases (Murdaugh et al., 1961). In terrestrial mammals that commonly breath-hold, such as the muskrat, there is no indication of an anticipatory ascent tachycardia (Drummond and Jones, 1979).

During the period of time when a diving mammal is breathing at the surface, vagal withdrawal in addition to elevation of sympathetic tone results in the elevated heart rate, which in turns helps to reduce the recovery time (Elliott et al., 2002; Signore and Jones, 1995). In addition, breathing also results in a respiratory tachycardia, helping to increase cardiac output (Blawas et al., 2021a,b; Cauture et al., 2019; Fahlman et al., 2020b, 2019). The northern elephant seal (*Mirounga angustirostris*) has an extremely high surface tachycardia, which allows it to spend a minimum of time at the surface between dives (Andrews et al., 1997). A preparatory increase in heart rate sometimes also occurs in voluntarily diving muskrats (Signore and Jones, 1995) immediately before a dive. This anticipatory elevation in heart rate is absent in muskrats treated with the adrenergic blockers propranolol and nadolol, suggesting that increased sympathetic tone is responsible for the increase.

Anticipatory changes in diving birds

During the vast majority of natural dives in birds, as in mammals, metabolism is largely – if not completely – aerobic, with the active skeletal muscles as well as the brain and heart receiving an adequate supply of O_2 (Fig. 3). Thus, during most natural dives, stored O_2 is used at an elevated rate compared with that of resting animals and is quickly replaced upon surfacing. Birds can perform dives in quick succession. However, in tufted ducks (*Aythya fuligula*) at the same level of aerobic metabolism, heart rate is lower during natural dives than during surface swimming. This led to the suggestion that the cardiovascular response to natural submersion is a balance between the Irving–Scholander ('classical') dive response (leading to bradycardia) and the response to exercise (stimulating tachycardia), with the bias towards the latter (Butler, 1985; Eliassen, 1960; Millard et al., 1973). This balance can be shifted in the direction of the response to involuntary submersion (i.e. showing a strong bradycardia) when the birds swim long horizontal distances or if they are briefly unable to surface. For instance, when tufted ducks swim long horizontal distances underwater for their food, heart rate progressively declines, such that by ∼30 s there is a significant bradycardia (Stephenson et al., 1986). If ducks are temporarily prevented from surfacing from a natural dive, there is an immediate reduction in heart rate similar to that seen during involuntary submersion (Stephenson et al., 1986). Thus, although extremely low heart rates have been reported in free-diving seals (Andrews et al., 1997; Thompson and Fedak, 1993), in birds a significant bradycardia has only been measured in the emperor penguin, during forced submersion or exclusion studies (i.e. studies where birds are suddenly prevented from reaching the surface; Butler, 1982; Butler and Jones, 1968; Folkow et al., 1967; Jones et al., 1988; Jones and Holeton, 1972; Ponganis and Kooyman, 2000; Stephenson et al., 1986). This led Furilla and Jones (1987) to suggest that there is a considerable cognitive component modulating cardiac responses in voluntarily diving birds. Thus, only during forced dives in restrained animals is cardiac control largely reflexogenic.

An exception to the pattern of diving in birds described above is the regulation of heart rate in spontaneously diving emperor penguins (*Aptenodytes forsteri*) (Fig. 4; Meir et al., 2008). In emperor penguins, a true bradycardia occurs during prolonged dives (i.e. dives greater than the aerobic dive limit). During such dives, heart rate declines significantly with increasing dive duration, independent of swim stroke frequency, suggesting that progressive bradycardia and peripheral vasoconstriction (including isolation of muscle) occur to conserve blood O_2 (Meir et al., 2008). Following these dives, heart rate is significantly elevated, presumably to enhance O_2 loading and repay an O_2 debt (see Glossary). Meir et al. (2008) concluded that the classic

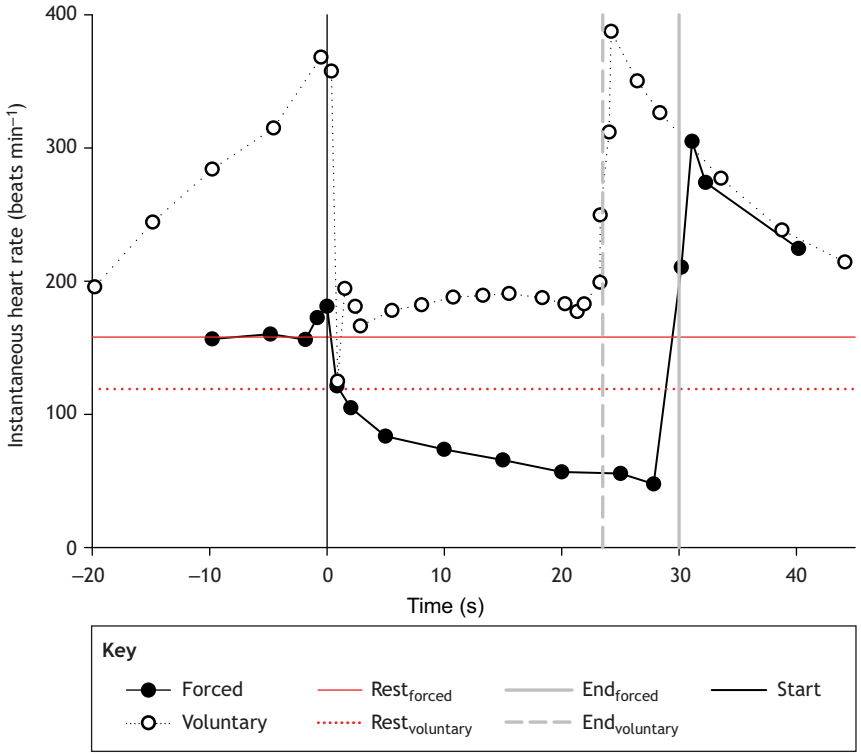

Fig. 3. Instantaneous heart rate as a function of time in diving tufted ducks (*Aythya fuligula*). Filled circles show a forced dive, in which there is a significant (progressive) bradycardia. Open symbols show a voluntary dive in which there is no diving bradycardia, or any progressive change in diving heart rate. In both dives, there is an anticipatory pre-dive tachycardia. Data redrawn from Butler (1988). The red horizontal lines indicate the average heart rate before diving in each case. The vertical lines indicate the beginning and end of the dives in each scenario.

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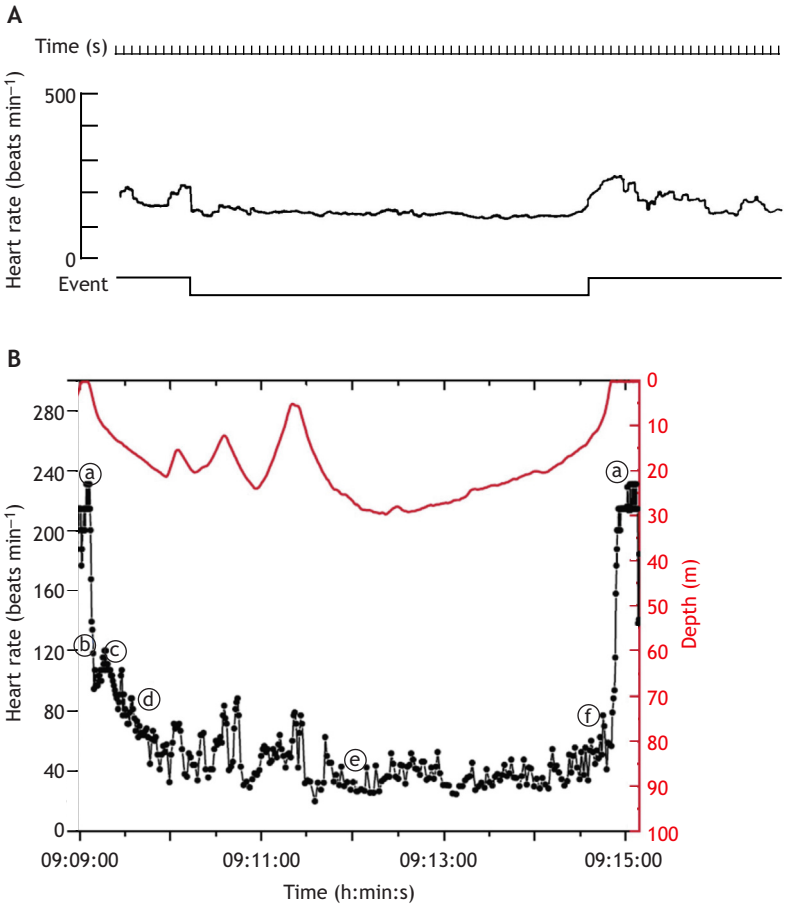

Fig. 4. Instantaneous heart rate as a function of time in freely diving penguins. (A) Humboldt penguin (*Spheniscus humboldti*). The period of submergence is indicated by a downward deflection of the event marker. (B) Emperor penguin (*Aptenodytes forsteri*). In this panel, dive depth is also shown. a, surface interval tachycardia (pre and post-dive); b, immediate heart rate decline on submergence; c, readjustment of heart rate; d and e, secondary successive bradycardia; f, ascent tachycardia. The Humboldt penguins weighed 4.3–5.9 kg, and the average body mass of the emperor penguins was 25.1 kg. The aerobic dive limit in the Humboldt penguin has been estimated as 1.9–2.3 min (Butler and Woakes, 1984; Luna-Jorquera and Culik, 2000), and in the emperor penguin it is 5–7 min (Ponganis et al., 1997a). Data redrawn from Butler and Woakes (1984) and Meir et al. (2008) for the Humboldt and emperor penguins, respectively.

Scholander–Irving dive response in emperor penguins contrasts with the absence of true bradycardia in diving ducks, cormorants (*Phalacrocorax auritus*), and king (*Aptenodytes patagonicus*), gentoo, Adélie and Humboldt (*Spheniscus humboldti*) penguins (Fig. 4; Butler and Woakes, 1984; Enstipp et al., 2001; Froget et al., 2004; Millard et al., 1973). It should be noted that an 'ascent' or 'anticipatory' tachycardia is also a common feature of most dives in emperor penguins as well as during prolonged dives in several penguin species and in ducks (Figs 3 and 4) (Froget et al., 2004; Green et al., 2003; Millard et al., 1973).

There are several studies showing that the dive response in birds can be conditioned. For example, the heart rate response in forced-diving pekin ducks (*Anas ptutyrhyngus*) and double-crested cormorants (*Phalacrocorax auritus*) is reduced during repetitive diving (Gabrielsen, 1985; Kanwisher et al., 1981). Gabbott and Jones (1987), studying both dabbling and diving avian species, showed that the onset of pronounced bradycardia is delayed with each successive trial, until eventually little to no bradycardia occurs during subsequent dives. In the dabbler, diving bradycardia is driven by chemoreceptors, but the changes in blood gases during dives performed by naive and habituated dabbling (*Anas platyrhynchos*) and diving redhead (*Aythya americana*) ducks are the same. This indicates that, in the absence of a demonstrated decrement in chemoreceptor sensitivity, the site of habituation must reside in the central nervous system (Gabbott and Jones, 1987).

Anticipatory changes in reptilian divers

The few studies that have examined heart rate in freely diving reptiles have shown that most reptiles also do not experience a dramatic bradycardia during routine dives. However, heart rates tend to decline modestly immediately upon submergence, and continue to fall during descent (Belkin, 1964; Butler et al., 1984; Gaunt and Gans, 1969; Heatwole et al., 1979; Jacob and McDonald, 1976; Southwood et al., 1999; Ware, 1993). An additional decrease in heart rate may occur if reptiles are disturbed during a dive (Heatwole et al., 1979; Smith et al., 1974). Furthermore, most reptiles increase their heart rate during ascent back to the surface. In the green sea turtle (*Chelonia mydas*), the pulmonary blood flow associated with breathing also increases as the animals swim to the surface to breathe (Butler et al., 1984). All of these changes, albeit modest, suggest that there is a cognitive component to the heart rate variation seen during diving in reptiles.

Anticipatory adjustments to cardiovascular function during diving in reptiles – or at least adjustments that are hard to attribute to simple reflexes – have also been inferred from rapid patterns of increase and decrease in arterial blood oxygenation. In the red-eared slider (*Trachemys scripta elegans*, formerly *Pseudemys scripta*), short (5–20 min) voluntary dives are characterized by alveolar gas P_{O_2} (PA_{O_2}) and arterial blood P_{O_2} (Pa_{O_2}) generally declining together in parallel from the initiation of the dive (Burggren and Shelton, 1979; Fig. 5). However, a second, less-common pattern of change in oxygenation is associated with voluntary, infrequent dives of long duration (e.g. >2 h). The first hour of these longer dives is characterized by a steep decline in Pa_{O_2} without an accompanying change in PA_{O_2}. After 40–80 min, however, PA_{O_2} suddenly drops steeply, concurrent with an actual rise in Pa_{O_2} that can take 10–30 min. After this point, PA_{O_2} levels off and Pa_{O_2} decreases sharply again. The physiological interpretation of these changes in

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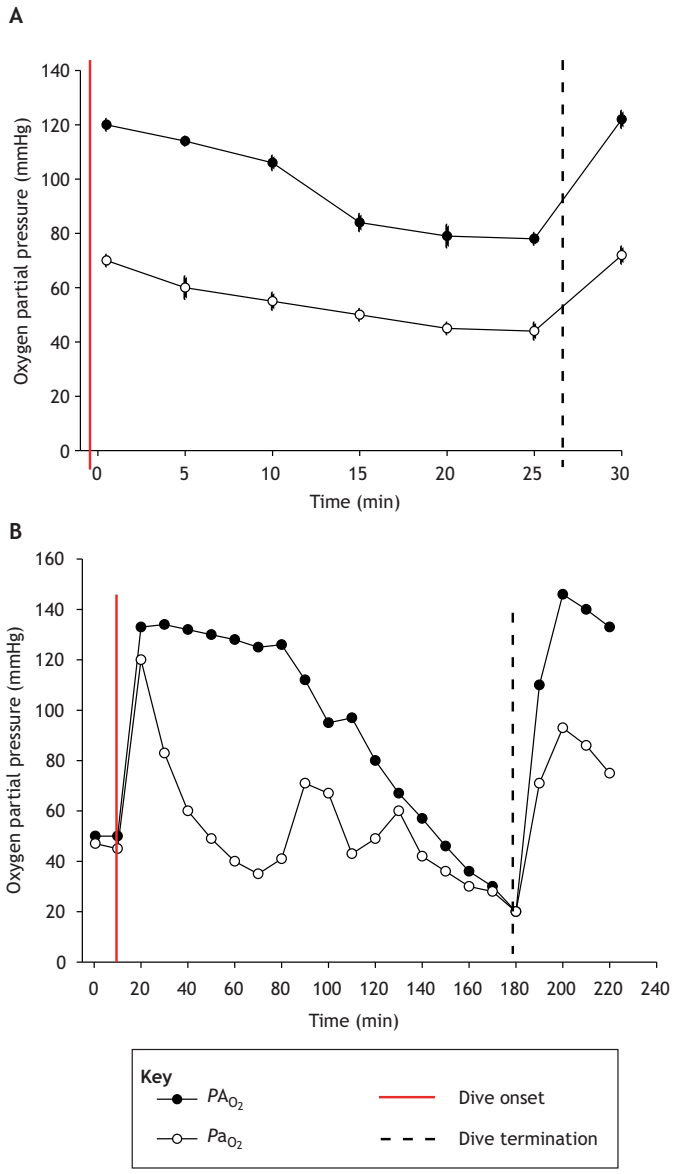

Fig. 5. Changes in lung O_2 partial pressure (PA_{O_2}) and femoral arterial blood O_2 partial pressure (Pa_{O_2}) during voluntary diving in the turtle (*Trachemys scripta elegans*). (A) Mean values±s.e.m. of O_2 levels typical of short-duration voluntary dives (n=8). (B) Changes in PA_{O_2} and Pa_{O_2} in an individual turtle during an atypically long voluntary dive of nearly 3 h, followed by the onset of a typical shorter dive. Note that in the short dives (A), PA_{O_2} and Pa_{O_2} decrease in parallel as diving progresses. In the longer dive (B), presumably a nearly complete right-to-left shunt develops for the first hour of the dive, preventing the transfer of O_2 from lung gas to arterial blood. Subsequently, however, blood is 'pulsed' through the lung, leading to multiple bouts of lung O_2 extraction and transfer to arterial blood. Importantly, these physiological patterns associated with either a short or long dive are evident in the very first minutes of the dive, suggesting some degree of cognitive control associated with 'planning' the future dive. Redrawn from Burggren and Shelton (1979).

oxygenation is that at the beginning of these long dives, the turtle effects an almost complete 'right-to-left shunt', in which blood returning to the heart bypasses the lungs and returns to the systemic circulation. This prevents the transfer of alveolar O_2 into pulmonary capillary blood. Consequently, Pa_{O_2} drops more steeply than it does during short voluntary dives, during which O_2 extraction from the lungs occurs as soon as the dive starts. Later in the long dives, PA_{O_2} plummets as the right-to-left shunt is reduced, allowing temporary transfer of alveolar O_2 into pulmonary venous and ultimately systemic arterial blood. This 'pulsing' of blood flow to the lungs presumably occurs to meter out alveolar O_2 stores. Importantly – and pertinent to the point of cognition and anticipation – turtles do not switch between the short-dive and long-dive patterns of blood flow and O_2 utilization during any single dive. Rather, they have apparently determined the period of the voluntary dive at its very outset. Although heart rate was not measured during these long dives, presumably there are changes in heart performance, if not actual heart rate – this is an area that would benefit from further research.

Conditioned changes in heart rate – potential mechanisms

Before we begin to consider potential mechanisms that might underlie volitional or conditioned changes in heart rate, it is important to consider what is known about the means by which the nervous system affects the function of the cardiovascular system. Cardiac function is controlled by either changes in contractility (inotropic changes) or changes in heart rate (chronotropic changes), both of which alter cardiac output. Chronotropic changes involve autonomic action on nerves of the sinoatrial node, the atrioventricular node and the ventricle. Furthermore, studies on both animal and human subjects have shown that operant conditioning can produce conditioned or volitional changes that are both inotropic and chronotropic in nature (Engel, 1972; Engel and Gottlieb, 1970). Moreover, pharmacological intervention has shown that the vagus nerve (acting on the atrioventricular node) is responsible for both acceleration and deceleration of heart rate (see references in Engel, 1972). By contrast, vascular tone is controlled by the sympathetic adrenergic and parasympathetic cholinergic branches of the autonomic nervous system. It is the sympathetic system that is responsible for the vasoconstriction of blood vessels associated with the diving response.

Accentuated antagonism or autonomic conflict

The dive response reflects a balance of autonomic function that produces changes in heart rate and vascular tone. However, this response also alters normal homeostatic reflexes, including the baroreceptor reflex and respiratory chemoreceptor reflex (see Glossary). The neurons driving the reflex circuits for these changes are contained within the medulla and spinal cord, because the relevant responses remain after brainstem transection at the pontomedullary junction (Panneton and Gan, 2020). Paradoxically, both sympathetic and parasympathetic activation are required for the diving response (the sympathetic system to bring about vasoconstriction and the parasympathetic system to reduce the heart rate), yet each has opposing effects on heart rate. During diving, a strong parasympathetic vagal tone decreases heart rate and prevents or minimizes sympathetic control of heart rate (Elliott et al., 2002; Levy, 1971; Signore and Jones, 1995). This 'accentuated antagonism' (see Glossary) results from a blunted sensitivity of cardiac cells to adrenergic stimulation during vagal activity. Thus, even weak vagal activity prevents cardiac acceleration, even in response to strong sympathetic stimulation (Kimura et al., 1985; Levy, 1971; Signore and Jones, 1995). Parasympathetic (vagal) control of heart rate can also occur more rapidly than changes produced by increased sympathetic tone (Akselrod et al., 1985; Japundzic et al., 1990; Signore and Jones, 1995). Consequently, accentuated antagonism, in which heart rate changes are made by altering the vagal tone, fosters rapid variation in heart rate. Although vagal tone suppresses the sympathetic cardiac accelerator fibres, the sympathetic vasomotor fibre tone maintains vasoconstriction (Murdaugh et al., 1968; Signore and Jones, 1995). This autonomic

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conflict (see Glossary) has also been posited as the source of the arrythmias sometimes seen in marine mammals during, for example, diving or sleep apnea (Ponganis et al., 2017).

Adjustments in perfusion during a dive appear to be governed by alterations in parasympathetic tone (Elliott et al., 2002; Signore and Jones, 1995). The area of the brain that provides central command and volitional control (the higher cortex) also modulates the activity of the parasympathetic system and regulates the degree of bradycardia at any point during a dive (Elmegaard et al., 2016; Elsner, 1965; Elsner et al., 1966b; Fahlman et al., 2020a; Fedak et al., 1988; Grinnell et al., 1942; Jobsis et al., 2001; Jones et al., 1973; Kaczmarek et al., 2018; Kooyman and Campbell, 1972; Ridgway et al., 1975). Thus, it is likely that we can never rule out a role for volitional control in heart rate regulation during diving in marine mammals, no matter how sophisticated the analysis. The use of such volitional control could be associated with higher-level perception of factors like risk (e.g. predation, being trapped under ice) or social interaction with conspecifics.

Volitional heart rate control and the selective gas exchange hypothesis

Decompression sickness (or the bends), is a series of symptoms resulting from the formation of gas emboli in the blood. During exposure to increasing pressure during descent of a diving animal, elevated levels of N_2 enter the blood. As the animal ascends and the pressure decreases (decompression), this N_2 comes out of solution (Fahlman, 2017; Fahlman et al., 2021). Scholander (1940) commented that the anatomy of the respiratory system in marine mammals, with a flexible chest and lung, and a stiff conducting airway, would result in alveolar compression and collapse during diving, with lung gas being pushed into the conducting airways. This reduces the uptake of N_2 and the risk of the bends, and is referred to as the balloon–pipe alveolar collapse model. However, neither theoretical studies using this model (which relies entirely on passive compression of the alveoli), nor empirical data can explain how marine mammals are able to prevent the formation of gas emboli during ascent (see fig. 3 in Fahlman et al., 2021).

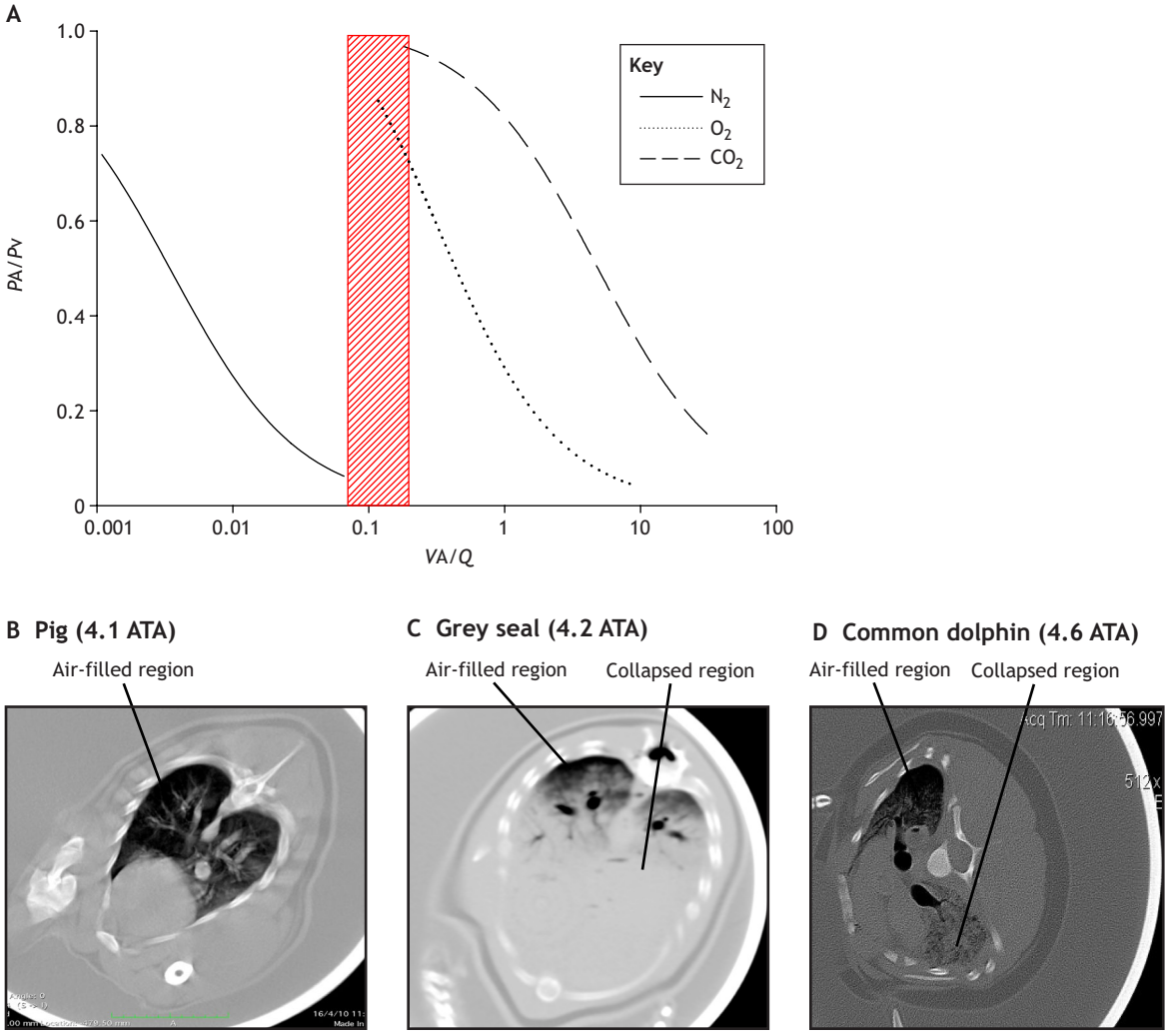

Fig. 6. Differences in gas solubility alter the pulmonary shunt as the ventilation-perfusion ratio varies, allowing selective gas exchange. (A) The pulmonary shunt (*PA*/*Pv*) varies from perfect gas equilibrium (1.0) to complete cessation of gas exchange (0) with ventilation–perfusion ratio (*VA*/*Q*; see Glossary) for gases with different gas solubility. The red box shows *VA*/*Q*, where there is exchange of O_2 and CO_2 with minimal or no exchange of N_2. (B–D) Computed tomography images show that different respiratory architectures result in differences in compression with increasing pressure, in the pig (B), the grey seal (C) and the common dolphin (D). In B–D, image slices show the anatomy at the mid-thoracic level behind the tracheal bifurcation; dark regions are air filled, whereas light grey regions are tissue. Reproduced from Fahlman (2024) and García-Párraga et al. (2018) under a creative commons license (http://creativecommons.org/licenses/by/4.0/). ATA, atmosphere absolute where 1 ATA is equal to 98.1 kPa.

The selective gas exchange hypothesis, however, provides a framework for how marine mammals and sea turtles may be able to selectively exchange O_2 and CO_2 at the surface, while minimizing or excluding the exchange of N_2 (Fahlman, 2024; Fahlman et al., 2021; García-Párraga et al., 2018). This hypothesis is based on empirical studies where gas elimination was measured in standing humans, where there is a gradient of ventilation–perfusion ratio in different regions of the lung, resulting in a pulmonary shunt (Farhi, 1967). Fig. 6A summarizes this concept, showing different levels of pulmonary shunt (PA/Pv, Fig. 6A), where a value of 1 represents perfect matching, in which the pulmonary capillary tension (Pv) and alveolar partial pressure are equal, and a value of 0 represents no gas exchange. From this figure, originally presented by Farhi (1967), we can see that the lung can act as a selective 'filter' depending on ventilation of the alveolus (VA) and pulmonary perfusion (Q). Thus, the relationship between VA/Q and pulmonary shunt varies considerably for gases with high solubility, e.g. O_2 and CO_2, as compared with N_2, which has low solubility (Fahlman, 2017, 2024). This hypothesis has been empirically tested in human subjects (West, 1962), where there are regional differences in the VA/Q ratio throughout the lung, which results in differences in gas exchange.

In diving animals, the lung is, of course, not ventilated during the dive, but it does contains gas; gas exchange therefore continues in the absence of ventilation (Burggren and Shelton, 1979; Fahlman et al., 2009; Kooyman et al., 1972; Kooyman and Sinnett, 1982; Patrician et al., 2021). Thus, in the following scenario, we assume that an alveolus that is not collapsed, and contains a mixture of O_2, CO_2 and N_2, is ventilated. As the diver descends, the partial pressure of gas increases, which initially increases diffusion. The increasing pressure also compresses gas-filled spaces, such as the alveoli, which results in a depth-related pulmonary shunt, due to the reduced VA and surface area. Eventually, the alveoli collapse and gas exchange ceases (Bostrom et al., 2008). In terrestrial mammals, this compression is similar to that of a balloon, and the chest and alveoli compress homogenously (see Fig. 6B, pig). In marine mammals, by contrast, alveolar compression is heterogenous, resulting in two distinct regions, one collapsed (light grey region) and the other open (dark region, see Fig. 6C,D, grey seal and common dolphin). In the collapsed region, there is no gas exchange and the VA/Q ratio is 0. In the open region, VA/Q is greater than 0; its exact value varies depending on the gas partial pressure, level of alveolar compression and perfusion to the alveoli. The latter can be varied by both anticipatory variation in heart rate (which modifies overall blood flow) and hypoxic pulmonary vasodilation (which alters the regional distribution of perfusion in the lung; Fahlman, 2024; Fahlman et al., 2021; García-Párraga et al., 2018). Thus, by varying the regional VA/Q ratio in open regions, it is possible to selectively exchange O_2 and CO_2 with minimal exchange of N_2 by selecting a VA/Q where PA/Pv is 1 for O_2 and CO_2, and 0 for N_2 (Fig. 6, shaded region). During normal diving, VA/Q is increased in part by the conditioned capacity to vary heart rate. During periods of stress (such as in response to sonar exposure in cetaceans), when sympathetic tone is increased, increasing perfusion to open regions results in a decreased VA/Q ratio, thus allowing increased exchange of N_2 and subsequent formation of gas emboli (Fahlman, 2017, 2024; Fahlman et al., 2021).

Conclusion

The dive response has been investigated for over a century, and considerable research has been dedicated to its potential involvement in conserving O_2 stores during a dive. 'Diving bradycardia' has been proposed as one of the hallmarks of the dive response. Yet, there is no clear quantitative relationship between dive capacity and heart rate reduction during diving (Mottishaw et al., 1999). Moreover, even terrestrial mammals experiencing voluntary or forced diving show a reduction in heart rate that is of a similar magnitude to that shown by some dive-adapted mammals during diving (Panneton, 2013). Thus, unravelling the evolutionary origins and selective forces behind diving bradycardia remains complex. By contrast, peripheral vasoconstriction and the maintenance of mean blood pressure appear to be physiological responses that are more well developed in diving vertebrates than in terrestrial mammals.

Many studies in dive-adapted vertebrates provide evidence that the dive response is not purely an autonomic reflex, but rather that it varies depending on a number of factors, can be conditioned and is likely to involve some degree of cognition. Dive-adapted animals show anticipatory heart rate control and probably have conditioned/volitional capacity to vary their heart rate depending on the planned dive, or to respond to changes in the dive plan. The variation in vagal tone that results from volitional control will produce benign arrythmias, as has been reported during both static and active dives in several species (Bickett et al., 2019; Fedak and Thompson, 1993). This physiological plasticity in perfusion allows the dynamic distribution of blood gases and offers dive-adapted species the capacity to vary both the temporal response and magnitude of the blood-flow changes, depending on the planned dive. Indeed, as suggested by Furilla and Jones (1987), it is only in forced dives performed by restrained animals, that cardiac control is largely reflexogenic (i.e. the 'classic diving response'); in freely diving animals there is considerable psychogenic (or cognitive) modulation of the diving response. Although such experiments are likely to be difficult, future work should aim to determine the role of cognition in the physiological changes associated with diving. The existence of a conditioned/volitional capacity to alter heart rate suggests that short-term habituation may be a simple method to mitigate sonar-related mass strandings of deep-diving whales; in addition, further study of the capacity to voluntarily alter heart rate may have implications for our understanding of the evolutionary physiology of cardiac function.

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Competing interests

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References

Akselrod, S., Gordon, D., Madwed, J. B., Snidman, N. C., Shannon, D. C. and Cohen, R. J. (1985). Hemodynamic regulation: investigation by spectral analysis. *Am. J. Physiol.* **249**, H867-H875.

Andersen, H. T. (1961). Physiological adjustments to prolonged diving in the American alligator, *Alligator mississippiensis*. *Acta Physiol. Scand.* **53**, 23-45. doi:10.1111/j.1748-1716.1961.tb02261.x

Andrews, R. D., Jones, D. R., Williams, J. D., Thorson, P. H., Oliver, G. W., Costa, D. P. and Le Boeuf, B. J. (1997). Heart rates of northern Elephant seals diving at sea and resting on the beach. *J. Exp. Biol.* **200**, 2083-2095. doi:10.1242/jeb.200.15.2083

Angell-James, J. E. and Daly, M. D. B. (1969). Cardiovascular responses in apnoeic asphyxia: role of arterial chemoreceptors and the modification of their effects by a pulmonary vagal inflation reflex. *J. Physiol.* **201**, 87-104. doi:10.1113/jphysiol.1969.sp008744

Angell-James, J. E., Elsner, R. and De Burgh Daly, M. (1981). Lung inflation: effects on heart rate, respiration, and vagal afferent activity in seals. *Am. J. Physiol. Heart Circ. Physiol.* **240**, H190-H198. doi:10.1152/ajpheart.1981.240.2.H190

Belkin, D. A. (1964). Variations in heart rate during voluntary diving in the turtle *Pseudemys concinna*. *Copeia* **1964**, 321-330. doi:10.2307/1441026

Bert, P. (1870). *Lecons sur la physiologie comparee de la respiration*. Paris: Bailiere.

Bickett, N. J., Tift, M. S., St. Leger, J. and Ponganis, P. J. (2019). Heart rates, heart rate profiles, and electrocardiograms in three killer whales, a beluga, and a pilot whale: an exploratory investigation. *Mar. Mamm. Sci.* **35**, 1112-1132. doi:10.1111/mms.12578

Blawas, A. M., Nowacek, D. P., Allen, A., Rocho-Levine, J. and Fahlman, A. (2021a). Respiratory sinus arrhythmia and submersion bradycardia in bottlenose dolphins (*Tursiops truncatus*). *J. Exp. Biol.* **224**, jeb234096. doi:10.1242/jeb.234096

Blawas, A. M., Nowacek, D. P., Rocho-Levine, J., Robeck, T. R. and Fahlman, A. (2021b). Scaling of heart rate with breathing frequency and body mass in cetaceans. *Philos. Trans. R. Soc. B: Biol. Sci.* **376**, 20200223. doi:10.1098/rstb.2020.0223

Blawas, A. M., Ware, K. E., Schmaltz, E., Zheng, L., Spruance, J., Allen, A. S., West, N., Devos, N., Corcoran, D. L., Nowacek, D. P. et al. (2021c). An integrated comparative physiology and molecular approach pinpoints mediators of breath-hold capacity in dolphins. *Evol. Med. Public Health* **9**, 420-430. doi:10.1093/emph/eoab036

Bostrom, B. L., Fahlman, A. and Jones, D. R. (2008). Tracheal compression delays alveolar collapse during deep diving in marine mammals. *Respir. Physiol. Neurobiol.* **161**, 298-305. doi:10.1016/j.resp.2008.03.003

Burggren, W. W. and Shelton, G. (1979). Gas Exchange and Transport During Intermittent Breathing in Chelonian Reptiles. *J. Exp. Biol.* **82**, 75-92. doi:10.1242/jeb.82.1.75

Burggren, W., Fahlman, A. and Milsom, W. (2024). Breathing patterns and associated cardiovascular changes in intermittently breathing animals: (Partially) correcting a semantic quagmire. *Exp. Physiol.* **109**, 1051-1065. doi:10.1113/EP091784

Butler, P. J. (1982). Respiratory and cardiovascular control during diving in birds and mammals. *J. Exp. Biol.* **100**, 195-221. doi:10.1242/jeb.100.1.195

Butler, P. J. (1985). The diving reflex in free-diving birds. In: *Arctic underwater operations. Medical and operational aspects of diving activities in arctic conditions*, (ed. L. Rey), pp. 49-61. Graham & Trotman Ltd.

Butler, P. J. (1988). The exercise response and the “classical” diving response during natural submersion in birds and mammals. *Can. J. Zool.* **66**, 29-39. doi:10.1139/z88-004

Butler, P. J. and Jones, D. R. (1968). Onset of and recovery from diving bradycardia in ducks. *J. Physiol.* **196**, 255-272. doi:10.1113/jphysiol.1968.sp008505

Butler, P. J. and Woakes, A. J. (1984). Heart rate and aerobic metabolism in Humboldt penguins, Spheniscus humboldti, during voluntary dives. *J. Exp. Biol.* **108**, 419-428. doi:10.1242/jeb.108.1.419

Butler, P. J., Milsom, W. K. and Woakes, A. J. (1984). Respiratory, cardiovascular and metabolic adjustments during steady state swimming in the green turtle, *Chelonia mydas*. *J. Comp. Physiol. B* **154**, 167-174. doi:10.1007/BF00684141

Casson, D. M. and Ronald, K. (1975). The harp seal, *Pagophilus groenlandicus* (Erxleben, 1777)—XIV. Cardiac arrythmias. *Comp. Biochem. Physiol. Part A: Physiol.* **50**, 307-314. doi:10.1016/0300-9629(75)90018-3

Cauture, F., SterbaBoatwright, B., Miedler, S., Rocho-Levine, J., Harms, C. and Fahlman, A. (2019). Using Respiratory Sinus Arrhythmia to estimate inspired tidal volume in the bottlenose dolphin (*Tursiops truncatus*). *Front. Physiol.* **10**, 128. doi:10.3389/fphys.2019.00128

Davis, R. W. and Kanatous, S. B. (1999). Convective oxygen transport and tissue oxygen consumption in Weddell seals during aerobic dives. *J. Exp. Biol.* **202**, 1091-1113. doi:10.1242/jeb.202.9.1091

DiCara, L. V. and Miller, N. E. (1968). Instrumental learning of systolic blood pressure responses by curarized rats: dissociation of cardiac and vascular changes. *Psychosom. Med.* **30**, 489-494. doi:10.1097/00006842-196809000-00002

Drummond, P. C. and Jones, D. R. (1979). The initiation and maintenance of bradycardia in a diving mammal, the muskrat, *Ondatra zibethica*. *J. Physiol.* **290**, 253-271. doi:10.1113/jphysiol.1979.sp012770

Eliassen, E. (1960). *Cardiovascularr Responses to Submersion Asphyxia in Avian Divers*, p. 100. Arbook University of Bergen.

Elliott, N. M., Andrews, R. D. and Jones, D. R. (2002). Pharmacological blockade of the dive response: effects on heart rate and diving behaviour in the harbour seal (*Phoca vitulina*). *J. Exp. Biol.* **205**, 3757-3765. doi:10.1242/jeb.205.23.3757

Elmegaard, S. L., Johnson, M., Madsen, P. T. and McDonald, B. I. (2016). Cognitive control of heart rate in diving harbor porpoises. *Curr. Biol.* **26**, R1167-R1176. doi:10.1016/j.cub.2016.10.020

Elsner, R. (1965). Heart rate response in forced versus trained experimental dives in pinnipeds. *Hvalrådets Skrifter* **48**, 24-29.

Elsner, R., Franklin, D. L., Van Citters, R. L. and Kenney, D. W. (1966a). Cardiovascular defense against asphyxia. *Science* **153**, 941-949. doi:10.1126/science.153.3739.941

Elsner, R., Kenney, D. W. and Burgess, K. (1966b). Diving bradycardia in the trained dolphin. *Nature* **212**, 407-408. doi:10.1038/212407a0

Engel, B. T. (1972). Operant conditioning of cardiac function: a status report. *Psychophysiology* **9**, 161-177. doi:10.1111/j.1469-8986.1972.tb00751.x

Engel, B. T. and Gottlieb, S. H. (1970). Differential operant conditioning of heart rate in the restrained monkey. *J. Comp. Physiol. Psychol.* **73**, 217-225. doi:10.1037/h0030217

Enstipp, M. R., Andrews, R. D. and Jones, D. R. (2001). The effects of depth on the cardiac and behavioural responses of double-crested cormorants (Phalacrocorax auritus) during voluntary diving. *J. Exp. Biol.* **204**, 4081-4092. doi:10.1242/jeb.204.23.4081

Fahlman, A. (2017). Allometric scaling of decompression sickness risk in terrestrial mammals; cardiac output explains risk of decompression sickness. *Scientific Rep.* **7**, 1-9. doi:10.1038/srep40918

Fahlman, A. (2024). Cardiorespiratory adaptations in small cetaceans and marine mammals. *Exp. Physiol.* **109**, 324-334. doi:10.1113/EP091095

Fahlman, A., Hooker, S. K., Olszowka, A., Bostrom, B. L. and Jones, D. R. (2009). Estimating the effect of lung collapse and pulmonary shunt on gas exchange during breath-hold diving: the Scholander and Kooyman legacy. *Respir. Physiol. Neurobiol.* **165**, 28-39. doi:10.1016/j.resp.2008.09.013

Fahlman, A., Miedler, S., Rocho-Levine, J., Jabois, A., Arenarez, J., Marti-Bonmati, L., García-Párraga, D. and Cauture, F. (2019). Re-evaluating the significance of the dive response during voluntary surface apneas in the bottlenose dolphin, *Tursiops truncatus*. *Sci. Rep.* **9**, 8613. doi:10.1038/s41598-019-45064-8

Fahlman, A., Cozzi, B., Manley, M., Jabas, S., Malik, M., Blawas, A. and Janik, V. M. (2020a). Conditioned variation in heart rate during static breath-holds in the bottlenose dolphin (*Tursiops truncatus)*. *Front. Physiol.* **11**, 604018. doi:10.3389/fphys.2020.604018

Fahlman, A., Miedler, S., Marti-Bonmati, L., Ferrero Fernandez, D., Muñoz Caballero, P., Arenarez, J., Rocho-Levine, J., Robeck, T. and Blawas, A. M. (2020b). Cardiorespiratory coupling in cetaceans; a physiological strategy to improve gas exchange? *J. Exp. Biol.* **223**, jeb226365. doi:10.1242/jeb.226365

Fahlman, A., Moore, M. J. and Wells, R. S. (2021). How do marine mammals manage and usually avoid gas emboli formation and gas embolic pathology? Critical clues from studies of wild dolphins. *Front. Mar. Sci.* **8**, 598633. doi:10.3389/fmars.2021.598633

Fahlman, A., Mcknight, J. C., Blawas, A. M., West, N., Torrente, A. G. and Aoki, K. (2023). Cardiorespiratory coupling in the bottlenose dolphin (*Tursiops truncatus*). *Front. Physiol.* **14**, 1234432. doi:10.3389/fphys.2023.1234432

Farhi, L. E. (1967). Elimination of inert gas by the lung. *Respir. Physiol.* **3**, 1-11. doi:10.1016/0034-5687(67)90018-7

Fedak, M. and Thompson, D. (1993). *Behavioural and Physiological Options in Diving Seals* (ed. I. L. Boyd), pp. 333-348. Clarendon Press.

Fedak, M. A., Pullen, M. R. and Kanwisher, J. (1988). Circulatory responses of seals to periodic breathing: heart rate and breathing during exercise and diving in the laboratory and open sea. *Can. J. Zool.* **66**, 53-60. doi:10.1139/z88-007

Folkow, B., Nilsson, N. J. and Yonce, L. R. (1967). Effects of diving on cardiac output in ducks. *Acta Physiol. Scand.* **70**, 347-361. doi:10.1111/j.1748-1716.1967.tb03634.x

Froget, G., Butler, P. J., Woakes, A. J., Fahlman, A., Kuntz, G., Le Maho, Y. and Handrich, Y. (2004). Heart rate and energetics of free-ranging king penguins (*Aptenodytes patagonicus*). *J. Exp. Biol.* **207**, 3917-3926. doi:10.1242/jeb.01232

Furilla, R. A. and Jones, D. R. (1987). The relationship between dive and pre-dive heart rates in restrained and free dives by diving ducks. *J. Exp. Biol.* **127**, 333-348. doi:10.1242/jeb.127.1.333

Gabbott, G. R. and Jones, D. R. (1987). Habituation of the cardiac response to involuntary diving in diving and dabbling ducks. *J. Exp. Biol.* **131**, 403-415. doi:10.1242/jeb.131.1.403

Gabrielsen, G. W. (1985). Free and forced diving in ducks: habituation of the initial dive response. *Acta Physiol. Scand.* **123**, 67-72. doi:10.1111/j.1748-1716.1985.tb07562.x

García-Párraga, D., Moore, M. and Fahlman, A. (2018). Pulmonary ventilation–perfusion mismatch: a novel hypothesis for how diving vertebrates may avoid the bends. *Proc. R. Soc. B* **285**, 20180482. doi:10.1098/rspb.2018.0482

Gaunt, G. S. and Gans, C. (1969). Diving bradycardia and withdrawal bradycardia in Caiman crocodilus. *Nature* **223**, 207-208. doi:10.1038/223207a0

Goldbogen, J. A., Cade, D. E., Calambokidis, J., Czapanskiy, M. F., Fahlbusch, J., Friedlaender, A. S., Gough, W. T., Kahane-Rapport, S. R., Savoca, M. S., Ponganis, K. V. et al. (2019). Extreme bradycardia and tachycardia in the world’s largest animal. *Proc. Natl. Acad. Sci. USA* **116**, 25329-25332. doi:10.1073/pnas.1914273116

Green, J. A., Butler, P. J., Woakes, A. J. and Boyd, I. L. (2003). Energetics of diving in macaroni penguins. *J. Exp. Biol.* **206**, 43-57. doi:10.1242/jeb.00059

Grinnell, S. W., Irving, L. and Scholander, P. F. (1942). Experiments on the relation between blood flow and heart rate in the diving seal. *J. Cell. Comp. Physiol.* **19**, 341-350. doi:10.1002/jcp.1030190309
Harrison, R. J. and Tomlinson, J. D. W. (1960). Normal and experimental diving in the common seal (*Phoca vitulina*). *Mammalia* **24**, 386-399.
Harrison, R. J., Ridgway, S. H. and Joyce, P. L. (1972). Telemetry of heart rate in diving seals. *Nature* **238**, 280-280. doi:10.1038/238280a0
Heatwole, H., Seymour, R. S. and Webster, M. E. D. (1979). Heart rates of sea snakes diving in the sea. *Comp. Biochem. Physiol. Part A: Physiol.* **62**, 453-455. doi:10.1016/0300-9629(79)90085-9
Hill, R. D. (1986). Microcomputer monitor and blood sampler for free-diving Weddell seals. *J. Appl Physiol (1985)* **61**, 1570-1576. doi:10.1152/jappl.1986.61.4.1570
Houser, D. S., Dankiewicz-Talmadge, L. A., Stockard, T. K. and Ponganis, P. J. (2010). Investigation of the potential for vascular bubble formation in a repetitively diving dolphin. *J. Exp. Biol.* **213**, 52-62. doi:10.1242/jeb.028365
Irving, L. (1963). Bradycardia in human divers. *J. Appl. Physiol.* **18**, 489-491. doi:10.1152/jappl.1963.18.3.489
Irving, L., Scholander, P. F. and Grinnell, S. W. (1941a). The respiration of the porpoise, *Tursiops truncatus*. *J. Cell. Comp. Physiol.* **17**, 145-168. doi:10.1002/jcp.1030170203
Irving, L., Scholander, P. F. and Grinnell, S. W. (1941b). Significance of the heart rate to the diving ability of seals. *J. Cell. Comp. Physiol.* **18**, 283-297. doi:10.1002/jcp.1030180302
Irving, L., Scholander, P. F. and Grinnell, S. W. (1942). The regulation of arterial blood pressure in the seal during diving. *Am. J. Physiol. Legacy Content* **135**, 557-566. doi:10.1152/ajplegacy.1942.135.3.557
Jacob, J. S. and McDonald, H. S. (1976). Diving bradycardia in four species of North American aquatic snakes. *Comp. Biochem. Physiol. Part A: Physiol.* **53**, 69-72. doi:10.1016/S0300-9629(76)80013-8
Japundzic, N., Grichois, M.-L., Zitoun, P., Laude, D. and Elghozi, J.-L. (1990). Spectral analysis of blood pressure and heart rate in conscious rats: effects of autonomic blockers. *J. Auton. Nerv. Syst.* **30**, 91-100. doi:10.1016/0165-1838(90)90132-3
Jobsis, P. D., Ponganis, P. J. and Kooyman, G. L. (2001). Effects of training on forced submersion responses in harbor seals. *J. Exp. Biol.* **204**, 3877-3885. doi:10.1242/jeb.204.22.3877
Johansen, K. (1959). Heart activity during experimental diving of snakes. *Am. J. Physiol. Legacy Content* **197**, 604-606. doi:10.1152/ajplegacy.1959.197.3.604
Jones, D. R. and Holeton, G. F. (1972). Cardiac output of ducks during diving. *Comp. Biochem. Physiol. A* **41**, 639-645. doi:10.1016/0300-9629(72)90018-7
Jones, D. R., Fisher, H. D., McTaggart, S. and West, N. H. (1973). Heart rate during breath-holding and diving in the unrestrained harbor seal. *Can. J. Zool.* **51**, 671-680. doi:10.1139/z73-101
Jones, D. R., Furilla, R. A., Heieis, M. R. A., Gabbott, G. R. J. and Smith, F. M. (1988). Forced and voluntary diving in ducks: cardiovascular adjustments and their control. *Can. J. Zool.* **66**, 75-83. doi:10.1139/z88-010
Kaczmarek, J., Reichmuth, C., McDonald, B. I., Kristensen, J. H., Larson, J., Johansson, F., Sullivan, J. L. and Madsen, P. T. (2018). Drivers of the dive response in pinnipeds; apnea, submergence or temperature? *J. Exp. Biol.* **221**, jeb176545. doi:10.1242/jeb.176545
Kanwisher, J., Gabrielsen, G. and Kanwisher, N. (1981). Free and forced diving in birds. *Science* **211**, 717-719. doi:10.1126/science.7192883
Kimura, T., Uchida, W. and Satoh, S. (1985). Predominance of postsynaptic mechanism in vagal suppression of sympathetic tachycardia in the dog. *J. Pharmacol. Exp. Therapy* **235**, 793-797.
Kooyman, G. L. (1985). Physiology without restraint in diving mammals. *Science* **1**, 166-178.
Kooyman, G. L. and Campbell, W. B. (1972). Heart rates in freely diving weddell seals, *Leptonychotes weddelli*. *Comp. Biochem. Physiol. Part A: Physiol.* **43**, 31-36. doi:10.1016/0300-9629(72)90465-3
Kooyman, G. L. and Sinnett, E. E. (1982). Pulmonary shunts in Harbor seals and sea lions during simulated dives to depth. *Physiol. Zool.* **55**, 105-111. doi:10.1086/physzool.55.1.30158447
Kooyman, G. L., Schroeder, J. P., Denison, D. M., Hammond, D. D., Wright, J. J. and Bergman, W. P. (1972). Blood nitrogen tensions of seals during simulated deep dives. *Am. J. Physiol.* **223**, 1016-1020. doi:10.1152/ajplegacy.1972.223.5.1016
Kooyman, G. L., Wahrenbrock, E., Castellini, M., Davis, R. and Sinnett, E. (1980). Aerobic and anaerobic metabolism during voluntary diving in Weddell seals: Evidence of preferred pathways from blood chemistry and behavior. *J. Comp. Physiol. B* **138**, 335-346. doi:10.1007/BF00691568
Kooyman, G. L., Castellini, M. A., Davis, R. W. and Maue, R. A. (1983). Aerobic diving limits of immature Weddell seals. *J. Comp. Physiol.* **151**, 171-174. doi:10.1007/BF00689915
Kooyman, G. L., McDonald, B. I., Williams, C. L., Meir, J. U. and Ponganis, P. J. (2020). The aerobic dive limit: after 40 years, still rarely measured but commonly used. *Comp. Biochem. Physiol. Part A: Mol. Integr. Physiol.* **252**, 110841. doi:10.1016/j.cbpa.2020.110841
Leivestad, H., Andersen, H. T. and Scholander, P. F. (1957). Physiological response to air exposure in codfish. *Science* **126**, 505. doi:10.1126/science.126.3272.505.a
Levy, M. N. (1971). Sympathetic parasympathetic interactions in the heart. *Circ. Res.* **29**, 437-445. doi:10.1161/01.RES.29.5.437
Lin, Y. C., Matsuura, D. T. and Whittow, G. C. (1972). Respiratory variation of heart rate in the California sea lion. *Am. J. Physiol. Legacy Content* **222**, 260-264. doi:10.1152/ajplegacy.1972.222.2.260
Luna-Jorquera, G. and Culik, B. M. (2000). Metabolic rates of swimming Humboldt penguins. *Mar. Ecol. Prog. Ser.* **203**, 301-309. doi:10.3354/meps203301
McDonald, B. I. and Ponganis, P. J. (2014). Deep-diving sea lions exhibit extreme bradycardia in long duration dives. *J. Exp. Biol.* **217**, 1525-1534. doi:10.1242/jeb.098558
McDonald, B. I., Johnson, M. and Madsen, P. T. (2018). Dive heart rate in harbour porpoises is influenced by exercise and expectations. *J. Exp. Biol.* **221**, jeb168740. doi:10.1242/jeb.168740
McDonald, B. I., Tift, M. S., Hückstädt, L. A., Jeffko, M. and Ponganis, P. J. (2020). Stroke effort and relative lung volume influence heart rate in diving sea lions. *J. Exp. Biol.* **223**, jeb214163. doi:10.1242/jeb.214163
McKnight, J. C., Bennett, K. A., Bronkhorst, M., Russell, D. J. F., Balfour, S., Milne, R., Bivins, M., Moss, S. E. W., Colier, W., Hall, A. J. et al. (2019). Shining new light on mammalian diving physiology using wearable near-infrared spectroscopy. *PLoS Biol.* **17**, e3000306. doi:10.1371/journal.pbio.3000306
McKnight, J. C., Patrician, A., Dujic, Z., Hooker, S. and Fahlman, A. (2024). Diving physiology in dolphins and human. In: *The Physiology of Dolphins* (ed. A. Fahlman and S. Hooker), pp. 135-146. Elsevier Press.
Meir, J. U., Stockard, T. K., Williams, C. L., Ponganis, K. V. and Ponganis, P. J. (2008). Heart rate regulation and extreme bradycardia in diving emperor penguins. *J. Exp. Biol.* **211**, 1169-1179. doi:10.1242/jeb.013235
Millard, R. W., Johansen, K. and Milsom, W. K. (1973). Radiotelemetry of cardiovascular responses to exercise and diving in penguins. *Comp. Biochem. Physiol. A Comp. Physiol.* **46**, 227-240. doi:10.1016/0300-9629(73)90414-3
Miller, N. E. (1969). Learning of visceral and glandular responses. *Science* **163**, 434-445. doi:10.1126/science.163.3866.434
Miller, N. E. and DiCara, L. (1967). Instrumental learning of heart rate changes in curarized rats: shaping, and specificity to discriminative stimulus. *J. Comp. Physiol. Psychol.* **63**, 12-19. doi:10.1037/h0024160
Mottishaw, P. D., Thornton, S. J. and Hochachka, P. W. (1999). The diving response mechanism and its surprising evolutionary path in seals and sea lions. *Am. Zool.* **39**, 434-450. doi:10.1093/icb/39.2.434
Murdaugh, H. V., Seabury, J. C. and Mitchell, W. L. (1961). Electrocardiogram of the Diving Seal. *Circ. Res.* **9**, 358-361. doi:10.1161/01.RES.9.2.358
Murdaugh, V. H., Jr., Cross, C. E., Millen, J. E., Gee, B. L. and Robin, E. D. (1968). Dissociation of bradycardia and arterial constriction during diving in the seal *Phoca vitulina*. *Science* **162**, 364-365. doi:10.1126/science.162.3851.364
Noren, S. R., Kendall, T., Cuccurullo, V. and Williams, T. M. (2012). The dive response redefined: underwater behavior influences cardiac variability in freely diving dolphins. *J. Exp. Biol.* **215**, 2735-2741. doi:10.1242/jeb.069583
Panneton, W. M. (2013). The mammalian diving response: an enigmatic reflex to preserve life? *Physiology* **28**, 284-297. doi:10.1152/physiol.00020.2013
Panneton, W. M. and Gan, Q. (2020). The mammalian diving response: inroads to its neural control. *Front. Neurosci.* **14**, 524. doi:10.3389/fnins.2020.00524
Patrician, A., Spajić, B., Gasho, C., Caldwell, H. G., Dawkins, T., Stembridge, M., Lovering, A. T., Coombs, G. B., Howe, C. A., Barak, O. et al. (2021). Temporal changes in pulmonary gas exchange efficiency when breath-hold diving below residual volume. *Exp. Physiol.* **106**, 1120-1133. doi:10.1113/EP089176
Ponganis, P. J. and Kooyman, G. L. (2000). Diving physiology of birds: a history of studies on polar species. *Comp. J. Biochem. Physiol. A.* **126**, 143-151. doi:10.1016/S1095-6433(00)00208-7
Ponganis, P. J., Kooyman, G. L., Starke, L. N., Kooyman, C. A. and Kooyman, T. G. (1997a). Post-dive blood lactate concentrations in emperor penguins, Aptenodytes forsteri. *J. Exp. Biol.* **200**, 1623-1626. doi:10.1242/jeb.200.11.1623
Ponganis, P. J., Kooyman, G. L., Winter, L. M. and Starke, L. N. (1997b). Heart rate and plasma lactate responses during submerged swimming and trained diving in California sea lions, *Zalophus californianus*. *J. Comp. Physiol. B* **167**, 9-16. doi:10.1007/s003600050042
Ponganis, P. J., McDonald, B. I., Tift, M. S. and Williams, C. L. (2017). Heart rate regulation in diving sea lions: the vagus nerve rules. *J. Exp. Biol.* **220**, 1372-1381. doi:10.1242/jeb.146779
Ridgway, S. H. and Howard, R. (1979). Dolphin lung collapse and intramuscular circulation during free diving: evidence from nitrogen washout. *Science* **206**, 1182-1183. doi:10.1126/science.505001
Ridgway, S. H., Carder, D. A. and Clark, W. (1975). Conditioned bardycardia in the sea lion *Zalophus californianus*. *Nature* **256**, 37-38. doi:10.1038/256037a0
Scholander, P. F. (1940). Experimental investigations on the respiratory function in diving mammals and birds. *Hvalrådets Skrifter* **22**, 1-131.
Scholander, P. F. (1963). The master switch of life. *Sci. Am.* **209**, 92-106. doi:10.1038/scientificamerican1263-92
Shearn, D. W. (1962). Operant conditioning of heart rate. *Science* **137**, 530-531. doi:10.1126/science.137.3529.530

Signore, P. E. and Jones, D. R. (1995). Effect of pharmacological blockade on cardiovascular responses to voluntary and forced diving in muskrats. *J. Exp. Biol.* **198**, 2307-2315. doi:10.1242/jeb.198.11.2307

Sinnett, E. E., Kooyman, G. L. and Wahrenbrock, E. A. (1978). Pulmonary circulation of the harbor seal. *J. Appl. Physiol.* **45**, 718-727. doi:10.1152/jappl.1978.45.5.718

Smith, E. N., Allison, R. D. and Crowder, W. E. (1974). Bradycardia in a free ranging American alligator. *Copeia* **1974**, 770-772. doi:10.2307/1442691

Sommer, L. S., McFarland, W. L., Galliano, R. E., Nagel, E. L. and Morgane, P. J. (1968). Hemodynamic and coronary angiographic studies in the bottlenose dolphin (*Tursiops truncatus*). *Am. J. Physiol.* **215**, 1498-1505. doi:10.1152/ajplegacy.1968.215.6.1498

Southwood, A. L., Andrews, R. D., Lutcavage, M. E., Paladino, F. V., West, N. H., George, R. H. and Jones, D. R. (1999). Heart rates and diving behavior of leatherback sea turtles in the Eastern Pacific Ocean. *J. Exp. Biol.* **202**, 1115-1125. doi:10.1242/jeb.202.9.1115cardic

Stephenson, R., Butler, P. J. and Woakes, A. J. (1986). Diving behaviour and heart rate in tufted ducks (*Aythya fuligula*). *J. Exp. Biol.* **126**, 341-359. doi:10.1242/jeb.126.1.341

Thompson, D. and Fedak, M. A. (1993). Cardiac responses of grey seals during diving at sea. *J. Exp. Biol.* **174**, 139-154. doi:10.1242/jeb.174.1.139

Vega, J. L. (2017). Edmund Goodwyn and the first description of diving bradycardia. *J. Appl. Physiol. (1985)* **123**, 275-277. doi:10.1152/japplphysiol.00221.2017

Ware, S. K. (1993). Cardiac responses of the turtle (*Trachemys scripta elegans)* to voluntary diving and to forced submersion. *Comp. Biochem. Physiol. Part A: Physiol.* **105**, 751-755. doi:10.1016/0300-9629(93)90279-D

Weingartner, G. M., Thornton, S. J., Andrews, R. D., Enstipp, M. R., Barts, A. D. and Hochachka, P. W. (2012). The effects of experimentally induced hyperthyroidism on the diving physiology of harbor seals (*Phoca vitulina*). *Front. Physiol.* **3**, 380. doi:10.3389/fphys.2012.00380

West, J. B. (1962). Regional differences in gas exchange in the lung of erect man. *J. Appl. Physiol.* **17**, 893-898. doi:10.1152/jappl.1962.17.6.893

Zapol, W. M., Liggins, G. C., Schneider, R. C., Qvist, J., Snider, M. T., Creasy, R. K. and Hochachka, P. W. (1979). Regional blood flow during simulated diving in the conscious Weddell seal. *J. Appl. Physiol.* **47**, 968-973. doi:10.1152/jappl.1979.47.5.968